Diagnostic Contributions of the Electromyogram

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SUMMARY

When muscles contract, they generate electricity. This muscle electricity can be seen and heard on the electromyograph. Normal muscles generate a different type of electrical impulse than do paralyzed muscles, and for this reason the electromyograph is of great assistance in the diagnosis, prognosis and treatment of such conditions as poliomyelitis, nerve injuries and strokes.

ELECTROMYOGRAPHY is the science which treats of the recording and measurement of the voltages generated by normal and abnormal muscles. In order to record and measure muscle voltages accurately, it is first necessary to elicit them from a muscle by means of a needle electrode in the muscle. Next these minute voltages must be amplified many times and then converted into visible patterns and sound energy by means of a cathode ray oscillograph and loud speaker. The instrument which elicits, amplifies, and converts the muscle voltages into visible patterns and sound energy is called an electromyograph (Figure 1) and the permanent record of the muscle voltages, which is obtained by photographing the visible patterns on the cathode ray oscillograph, is called an electromyogram. When interpreted correctly, electromyograms are a useful adjunct to the neurological examination in assessing the functional integrity of the neuromuscular system.

Relaxed normal voluntary muscle generates no voltages which can be recorded by the electromyograph. In other words, a completely relaxed normal voluntary muscle is an "electrically silent" one from the electromyographic standpoint. Normal voluntary muscle which is made to contract as a result of either voluntary or reflex effort generates what are termed "normal motor unit voltages."

A motor unit has been defined by Denny-Brown and Pennybacker[3] as a functional unit consisting of one anterior horn cell together with its axon and the 100 to 150 muscle fibers which the axon innervates. When the axon of such a motor unit is excited, all of the muscle fibers appear to contract synchronously, and as a result of the spread of the contraction wave, a simple (monophasic, diphasic, or triphasic) voltage is generated.

Normal motor unit voltages range in magnitude from about 100 to 2,000 microvolts and are readily elicited from all areas of the contracting normal muscle. The duration of a single wave is of the order of 3 to 10 milliseconds, and the repetition frequency varies from about 5 to 30 per second, depending on the force of contraction. Because of their simple wave form and relatively long duration, normal motor unit voltages produce a very characteristic thumping noise in the loud speaker.

When voluntary muscle has been deprived of its nerve supply or denervated for various periods of time, depending upon the species, the individual denervated muscle fibers begin to twitch in a rhythmic manner. This rhythmic twitching of denervated muscle has been termed denervation fibrillation and, according to Denny-Brown and Pennybacker,[3] results when the denervated muscle fibers become sensitized by neural atrophy to small amounts of acetylcholine in the normal circulation. Since the term denervation fibrillation denotes the involuntary contractions of individual muscle fibers, it is of great importance to note that true denervation fibrillation cannot be observed clinically through the intact skin. In order, therefore, to detect and prove its presence in a muscle, it is necessary to employ electromyography. The voltages generated by fibrillating denervated muscles are termed denervation fibrillation voltages and range in magnitude from about 5 to 100 microvolts. They usually have a diphasic wave form, and their repetition frequency varies from about 2 to 30 per second. A single wave has a duration of only 1 to 2 milliseconds, and because of its extremely short duration, a fibrillation voltage produces a very characteristic clicking noise in the loud speaker.

When a denervated muscle becomes reinnervated —that is, when it again acquires nerve substance through regeneration of its own nerve—it generates motor unit voltages which are highly polyphasic or complex in wave form. Because Weddell and associates[7] observed these highly complex motor unit voltages in muscles during the early stage of reinnervation after peripheral nerve lesions, they designated them as "nascent" motor unit action potentials or voltages. In a later communication, these investigators[8] stated that they believed the term "nascent" to be an inaccurate one because they thought it theoretically possible to have highly polyphasic or complex motor unit voltages during the course of demyelinating diseases. Other investigators[4] recently reported that complex motor unit voltages are a constant finding in muscles following poliomyelitis and in such degenerative diseases as amyotrophic lateral sclerosis and progressive muscular atrophy. From these observations, they have concluded that highly polyphasic or complex motor unit voltages are abnormal electromyographic

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phenomena and as such may represent either early nerve degeneration or early nerve regeneration.

It was pointed out previously that the muscle fibers of a normal motor unit contract synchronously, thus producing a simple motor unit voltage. The muscle fibers of an abnormal motor unit, on the other hand, appear to contract asynchronously, thus producing a complex or highly polyphasic motor unit voltage.

Complex or abnormal motor unit voltages range in magnitude from about 1,000 to 1,500 microvolts. Their repetition frequency varies from about 2 to 30 per second, and the duration of a single wave group is usually in the range of 5 to 15 milliseconds. Because of the complexity of these waves, abnormal motor unit voltages give rise to a very rough sounding noise in the loud speaker.

Most clinicians usually associate fibrillary twitchings of muscles with such degenerative diseases as amyotrophic lateral sclerosis and progressive muscular atrophy. The electromyographist, however, prefers to designate such visible involuntary contractions as fasciculations and to reserve the term denervation fibrillation for the invisible involuntary contractions of individual denervated muscle fibers. From the electromyographic standpoint, a complex fasciculation voltage simply represents the involuntary contraction of an abnormal motor unit. The characteristic voltages generated by normal, denervated and partially denervated muscles are illustrated in Figure 2.

It should be emphasized that denervation fibrillation is a phenomenon characteristic of denervated muscle, and for this reason denervation fibrillation voltages are not elicited from a muscle unless Wallerian degeneration of the nerve supply has taken place. Because of this fact, denervation fibrillation is a valuable objective sign of lower motor neuron disease. This is true because it is a constant finding following known lesions of the final common path and one that can neither be controlled by the patient nor observed clinically through the intact skin by the physician. When recorded electromyographically, however, its presence aids greatly in differentiating between upper and lower motor neuron disease,[1] myopathy and atrophy,[8] and functional and organic paralyses.[4] (Any precise objective test finds wide application in medicolegal fields, and electromyograms obtained by photographing a cathode ray oscillograph have been accepted as evidence in both federal and superior courts in California.)

The presence of denervation fibrillation can be of use in localization.[2,6] If for example, an upper extremity is examined and denervation fibrillation is noted only in those muscles receiving innervation from the median nerve, the electromyographic findings are consistent with a lower motor neuron lesion of the median nerve. In addition, it may be concluded that a certain percentage of the axons have undergone Wallerian degeneration. If on the other hand, the distribution of the denervation fibrillation corresponds to the distribution of the 7th cervical root, the electromyographic findings are consistent with a lower motor neuron lesion involving the 7th cervical root. Depending on the percentage of muscle fiber fibrillation, it can be further concluded that the electromyographic findings are consistent with the patient's complaints of paralysis, paresis, or fatigability. In order to express an opinion, however, concerning the cause of the peripheral nerve or root lesion, the electromyographic findings must

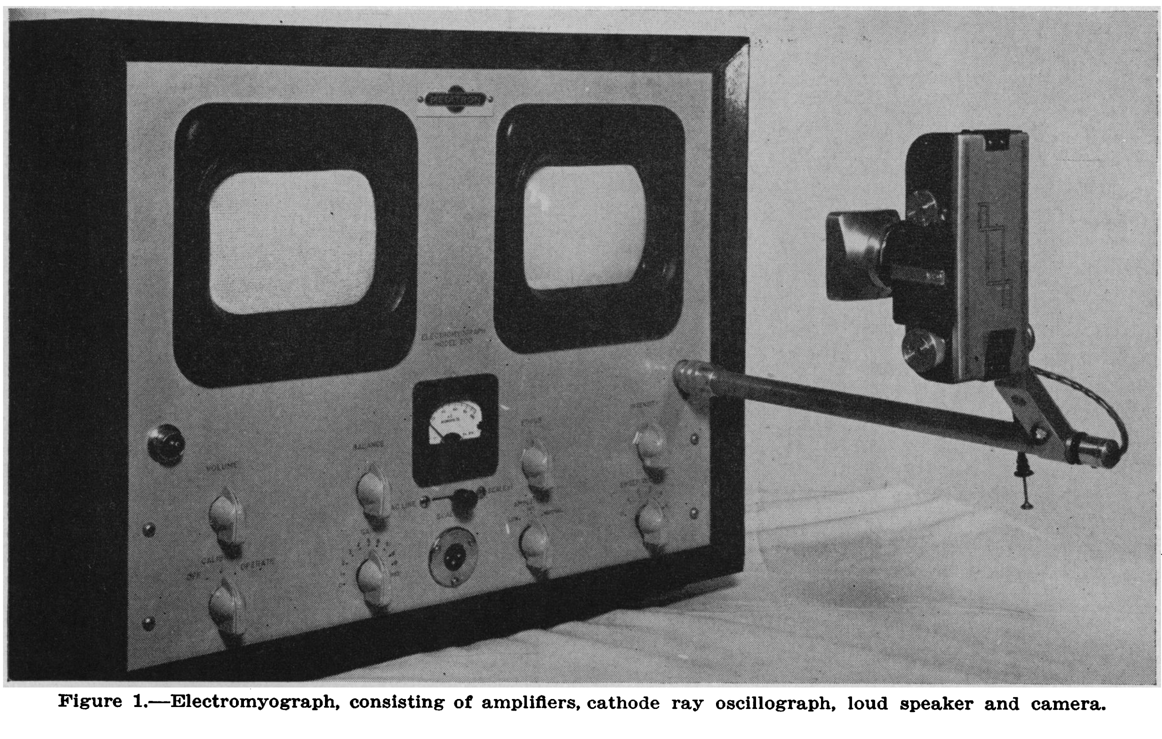

Figure 1.—Electromyograph, consisting of amplifiers, cathode ray oscillograph, loud speaker and camera.

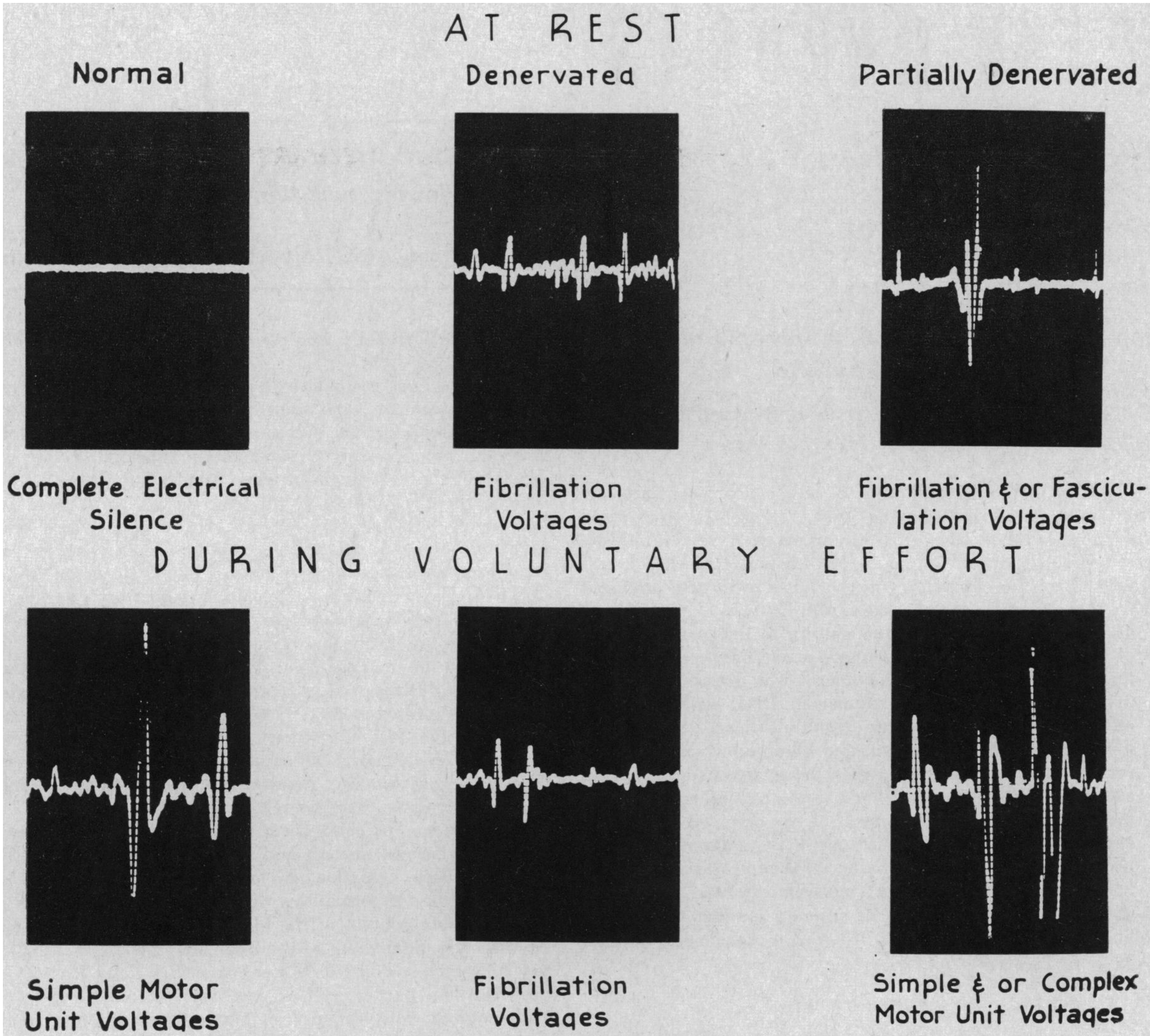

Figure 2.—Electromyograms, showing characteristic voltages of normal and abnormal voluntary muscle.

be correlated with a careful history and clinical examination.

While it is true that the presence of denervation fibrillation in a muscle indicates that some of the muscle fibers are completely denervated, it likewise indicates that these muscle fibers are still contractible tissue and capable of reinnervation once a nerve regenerates to them. This fact is very helpful in forming a prognosis relative to either ultimate functional recovery or ultimate permanent disability. When, however, an appreciable amount of denervation fibrillation in a muscle is noted, these muscles should receive vigorous physical treatment in the form of interrupted galvanic stimulation combined with heat and massage to prevent irreversible fibrotic changes and consequently a poor functional result.[5] A case in point is the patient with fracture and concomitant peripheral nerve lesions, who because of extensive wasting and fibrosis of the muscles is unable to use the injured limb even though the bones heal well.

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REFERENCES

1. Bauwens, P.: Electromyography in clinical medicine, The British J. of Phys. Med., May-June 1947.
2. Brazier, M. F. A., Watkins, A. L., and Michelsen, J. J.: Electromyography in differential diagnosis of ruptured cervical disk, Arch. Neurol. and Psychiat., 56:651, Dec. 1946.
3. Denny-Brown, D., and Pennybacker, J. B.: Fibrillation and fasciculation in voluntary muscle, Brain, 61:311, Sept.-Nov. 1938.
4. Golseth, J. G., and Huddleston, O. L.: Electromyographic diagnosis of lower motor neuron disease, Archives of Physical Medicine, 30:495, Aug. 1949.
5. Golseth, J. G., and Fizzell, J. A.: Electromyographic studies on cats after section and suture of the sciatic nerve, Amer. J. Physiol., 150:558, Oct. 1947.
6. Hoefer, P. F. A., and Guttman, S. A.: Electromyography as a method for determination of level of lesions in the spinal cord, Arch. Neurol. and Psychiat., 51:415, May 1944.
7. Weddell, G., Feinstein, B., and Pattle, R. E.: The clinical application of electromyography, Lancet, 1:236, Feb. 20, 1943.
8. Weddell, G., Feinstein, B., and Pattle, R. E.: The electrical activity of voluntary muscle in man under normal and pathological conditions, Brain, 67:178, Sept. 1944.